

GIANT CELLULAR MYOCARDITIS: WHICH STRATEGY SHOULD BE USED TO TREAT?



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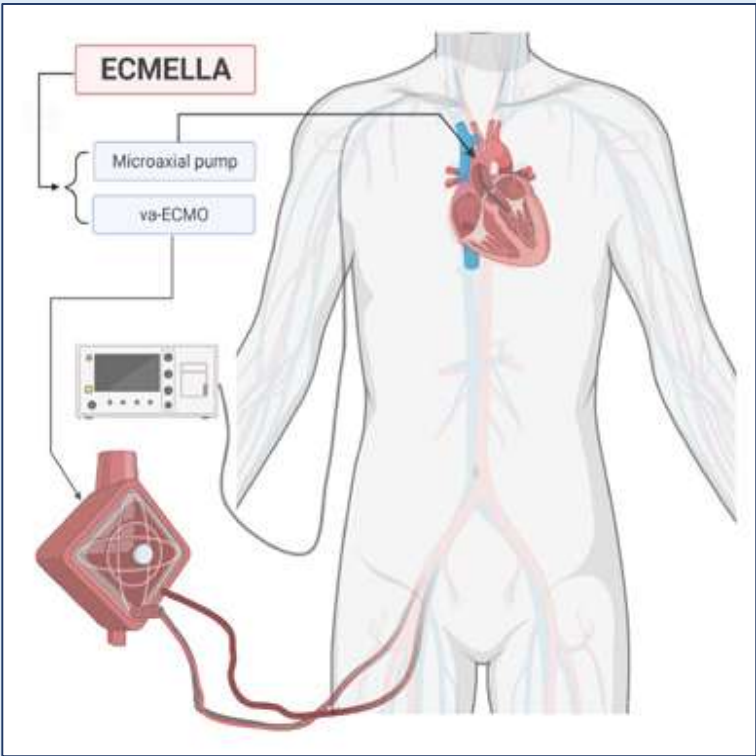
BACKGROUND

**Giant cell myocarditis (GCM)** is a rare disease histologically characterized by the presence of giant multinucleated cells along with extensive inflammatory infiltrates with large areas of necrosis. The pathophysiology is not well understood beyond its recognition as an autoimmune disorder attributable to T-lymphocyte-mediated myocardial inflammation. The prognosis is poor, and death often ensues unless a heart transplant is performed. The use of immunosuppressive therapy has significantly altered the prognosis of GCM.

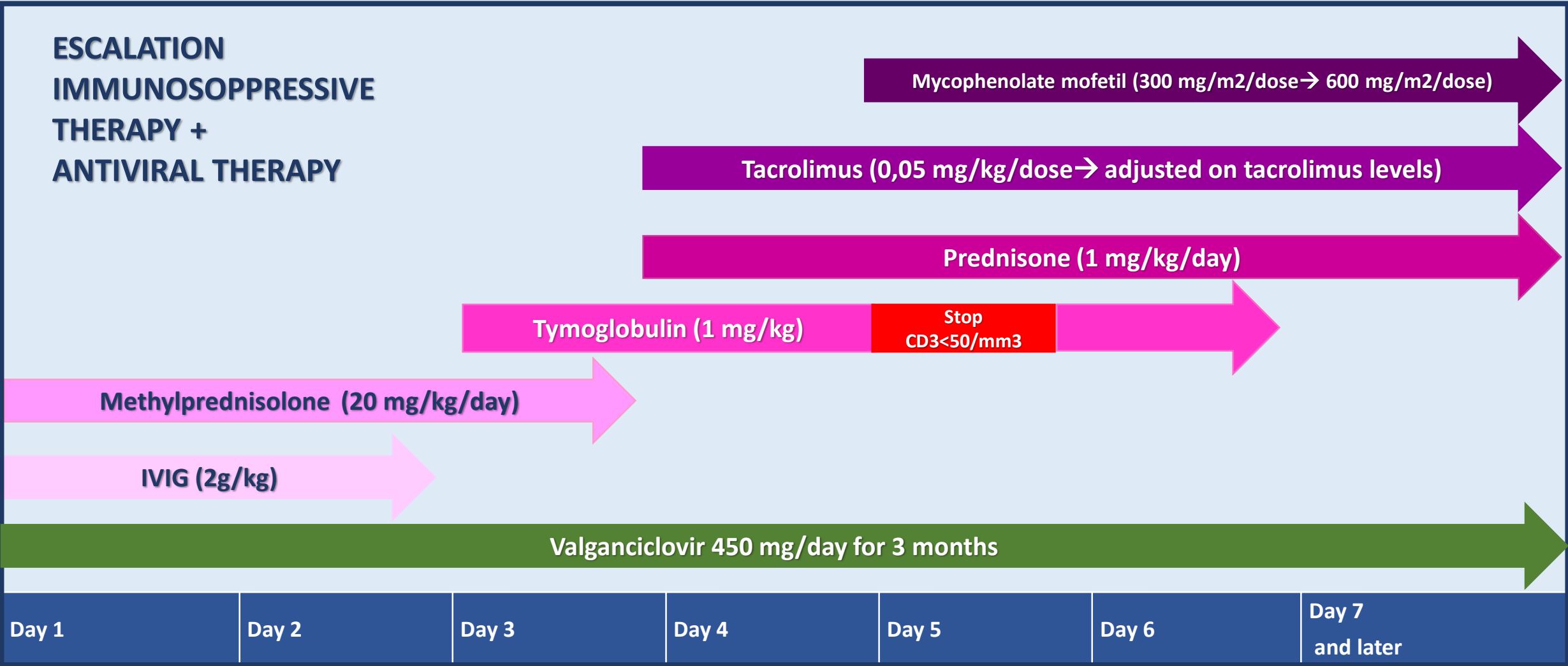
CASE REPORT

|                          |                          |
|--------------------------|--------------------------|
| Clinical signs           | pallor, cold extremities |
| Blood Pressure (mmHg)    | 75/55                    |
| Lactic acidosis (mmol/L) | 10                       |
| hsTnT (pg/ml)            | 1291                     |
| Nt-proBNP (pg/ml)        | 29000                    |
| Chest X-ray              | acute pulmonary oedema   |
| ECG                      | diffuse ST-segment ↓     |
| Echocardiogram           | LVEF 10%, severe MR      |

A 17-year-old girl presented with acute heart failure, rapidly evolving to cardiogenic shock. IV diuretics and adrenaline were started and mechanical circulatory support (MCS) devices with arteriovenous extracorporeal membrane oxygenation (ECMO) in addition to a femorally LV-aortic microaxial pump (Impella), also referred as **ECMELLA**, were placed achieving hemodynamic stabilization.



Endomyocardial biopsy (EMB) revealed an intense inflammatory infiltrate with **abundant multinucleated giant cells** as well as low-load Epstein Barr virus (200 copies/ml), Human Herpes virus-6 (<500 copies/ml) and Parvovirus B-19 virus (<100 copies/ml). **Combined immunosuppressive and antiviral therapy** was started.



A second EMB showed a **reduction in inflammatory infiltration**, without fibrosis. As the persistence of severe cardiac dysfunction we escalated MCS support to a surgical **LVAD Heart Mate III** as a bridge to recovery or alternatively, as bridge to heart transplantation.

CONCLUSION

Our case shows the importance of early recognition of myocarditis and timely initiation of therapy. The **combination of immunosuppressive and protective antiviral therapy along with MCS** was helpful in achieving hemodynamic stabilization, ventricular unloading and reduction of inflammatory infiltration in histology.